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Physiological Correlates of Stress-Induced Decrements in Human Perceptual Performance

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16. Abstract

Stress-induced changes in human performance have been thought to result from alterations in the "multidimensional arousal state" of the individual, as indexed by alterations in the physiological and psychological mechanisms controlling performance. Identification of such changes in substrate activities provide more complete descriptions of both the performance changes and the arousal state/mechanisms. In this study, decrements in perceptual performance were produced by independent and combined administration of atropine, sleep loss and exercise for both a visual aircraft identification task and an auditory vigilance task; measurements of performance changes were accompanied by state measures of cardiovascular function, pupillary diameter, sleep onset latency, and subject self-reports. Observed performance changes were accompanied by monotonic increases in heart rate after atropine and exercise, but not sleep loss. Moderate exercise produced blood pressure changes indicative of physical workload, but only atropine increased diastolic blood pressure and pupillary diameter relative to performance effects. Atropine and sleep loss each reduced sleep onset times to less than 50 percent control values ($p<.0001$); when combined with exercise, sleep onset times were reduced further ($p<.03$). These reductions in general arousal were confirmed by subject self-reports of reduced attentiveness and competence. These state measures of organismic function were found to be discriminatively correlative, but not predictive, of the decrements in perceptual performance seen; however, practical combinations of appropriate real-time measurement techniques could be developed that would promote the telemetering of human physiological activity to signal performance breakdown.

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PHYSIOLOGICAL CORRELATES OF STRESS-INDUCED DECREMENTS IN HUMAN PERCEPTUAL PERFORMANCE

INTRODUCTION

Effects of stress on human physiology and performance are generally accepted as resulting from changes in arousal (Broadbent, 1971; Hockey, 1979; Sanders, 1983). Included are changes in autonomic functions and behavioral efficiency. The pattern of observed responses induced by stressful conditions is thought to reflect changes in the "multidimensional arousal state" of the individual, as determined by effects on the balance of the underlying physiological and psychological arousal mechanisms (Beatty, 1986; Hamilton, Hockey & Rejman, 1977). Many stressors have been shown to selectively modulate only certain arousal mechanisms to produce discrete changes in behavioral functions, as opposed to effects on general arousal leading to global changes in performance (Pribram & McGuinness, 1976; Sanders, 1983, 1986; Tomparowski & Ellis, 1986; Warburton, 1975). Among these, atropine, sleep loss and exercise have been shown to selectively affect perceptual input processing, through apparent control of the "aperture" of perceptual apprehension. Low arousal provides a wide perceptual aperture, and thus a broadly distributed perceptual processing function; increases in arousal narrow the aperture to provide greater focusing (selectivity) of perceptual activity (Easterbrook, 1959). Atropine, sleep deprivation and exercise also produce discriminative changes in central and peripheral autonomic nervous system function (Weiner, 1980; Colquhoun, 1982; Fox, 1984). Because of this duality of selective effects, it was hypothesized that combined measurement of their singular and interactive effects on perceptual performance functions, accompanied by state measurements of their effects on autonomic functions, could begin to define the physiological basis of arousal-controlled alterations in perception. This knowledge could then provide a baseline for future real-time studies of the relationships between arousal, performance and autonomic physiology, and lead to potential discriminations about the cause(s) of functional disruptions.

METHODS

The protocols were chosen to replicate previously reported findings at higher atropine doses, using a visual aircraft identification task in a signal detection paradigm, and an auditory vigilance task employing five differently pitched tones of which the low tone was the target. The presented tones were grouped in 7.5 minute blocks of trials to allow for analysis of time-on-task effects. Sixty-four male volunteers, ranging in age from 21-35 years, participated in the study. All were in excellent health, weighed between 158 and 210 lbs, and all received a maximal exercise stress test administered according to the Bruce (1977) protocol. The research design employed an intromuscular atropine dose (2.0 mg or placebo) and prior moderate treadmill exercise as between-groups factors within experimental day; a night of sleep deprivation was the within-groups factor, counterbalanced by day. Two performance task cycles were accomplished each experimental day; each of these was preceded by the exercise treatment, which produced 75% of the subject's maximum heart rate. This procedure allowed two subjects, one drug and one placebo, to run in tandem. Both the exercise events and the task cycles were bracketed by state assessments of heart rate, systolic and diastolic blood pressure, and pupillary diameter. Thus, there were eight physiological measurement sessions each day. The drug injection occurred 15 minutes after the second exercise session, generally around noon. Subjects' self-perceptions were also recorded for items selected to generally correspond to four domains: alertness, attentiveness, competence, and comfort. These variables were chosen to provide additional subjective indications of stress effects, as well as lead to additional information about the changes in "multidimensional arousal state" produced by the stressors. The *Multiple Sleep Latency Test* (MSLT) (Carskadon & Dement, 1982) was administered three times during the day to provide a more direct measure of general arousal.

PERFORMANCE RESULTS

Aircraft Identification

The subjects' task was to detect the intermittent occurrences of friend or foe aircraft flying towards them on a visually "noisy" computer display screen, to be followed by a button press to "shoot down" the foes but not the friends. The response variables of interest were the signal detection statistics d-prime (stimulus sensitivity), hits (correct detections), false alarms (errors of commission), and beta (response bias). A four-way (drug x day x cycle x exercise) analysis of variance (ANOVA) was performed on the data, showing atropine main effects which reduced d-prime ($p < .01$) and hits ($p < .001$), and increased false alarms ($p < .03$); see Table 1. These results indicate decrements in stimulus sensitivity produced by atropine without effects on response bias. Sleep deprivation was also shown to cause reductions in d-prime ($p < .002$) and hits ($p < .001$), and increase false alarms ($p < .01$). It also produced a small but significant increase in beta scores ($p < .01$) for task cycle 1, which failed to replicate for task cycle 2. The interaction of atropine and sleep deprivation reduced hits further ($p < .01$), (Table 2). These effects suggest that in addition to reductions in perceptual sensitivity, subjects had difficulty sustaining attention; this tendency was seen particularly in the morning. There were no exercise main effects, although a further (nearly significant) reduction in hits for task cycle 1 ($p < .06$) was produced by the combination of exercise and sleep deprivation.

Auditory Vigilance

This task provided data consistent with those found for aircraft identification. A five-way (drug x day x cycle x block x exercise) ANOVA was performed on the data, signaling atropine effects on d-prime ($p < .008$) and hits ($p < .0001$), without effects on false alarms or beta. These effects were more pronounced on the sleep-deprived day, as evidenced by the (atropine x sleep deprivation) interaction effect ($p < .04$), on hits alone, (Table 3). A simple main effects analysis confirmed a relatively large decrease in hits in task cycle 2 on the sleep-deprived day ($p < .0001$). The effects of sleep deprivation were seen in a three-way (day x block x exercise) ANOVA on task cycle 1 scores for all subjects. Main effects of sleep deprivation were found on d-prime ($p < .001$), hits ($p < .0001$), and false alarms ($p < .02$). Again, these effects signal reduced perceptual sensitivity; however, the finding of increased beta ($p < .0004$), which was replicated in cycle 2 ($p < .02$), also indicates changes in response bias; i.e., subjects were generally not as responsive after sleep deprivation.

TABLE 1

Atropine Effects on Aircraft Identification

	D-prime		Hits		False Alarms	
Task Cycle	Atropine	Placebo	Atropine	Placebo	Atropine	Placebo
1	4.9	4.9	95.8	94.5	7.3	7.2
2	3.2	4.7	87.9	94.1	13.1	9.2

Response: Hits = percent hits False alarms = percent false alarms

TABLE 2

Atropine, Sleep Deprivation and Exercise Effects on Aircraft Identification

ATROPINE

Task Cycle		EX Day 1 X	EX Day 1 s_x	EX Day 2 X	EX Day 2 s_x	NEX Day 1 X	NEX Day 1 s_x	NEX Day 2 X	NEX Day 2 s_x
1	D-prime	5.5	0.5	3.7	1.8	5.9	2.2	4.4	2.1
	Hits	96.0	9.0	93.1	8.2	97.7	1.7	95.3	5.1
	FA's	6.7	5.3	10.2	6.1	4.5	3.3	7.8	7.1
	Beta	0.4	0.8	0.8	1.0	0.8	1.2	0.9	0.8
2	D-prime	4.2	2.7	2.2	1.7	4.2	2.4	2.4	0.9
	Hits	93.2	10.0	76.6	26.0	94.7	4.6	87.2	9.5
	FA's	9.4	7.5	18.2	13.3	9.5	9.8	15.3	12.5
	Beta	0.7	0.4	0.9	0.5	1.0	1.2	1.1	0.9

PLACEBO

Task Cycle		EX Day 1 X	EX Day 1 s_x	EX Day 2 X	EX Day 2 s_x	NEX Day 1 X	NEX Day 1 s_x	NEX Day 2 X	NEX Day 2 s_x
1	D-prime	5.3	2.6	4.3	2.4	5.6	2.7	4.5	2.5
	Hits	96.9	3.8	91.3	3.8	91.3	10.9	93.9	7.6
	FA's	6.0	5.6	6.5	6.9	7.8	10.8	8.4	2.6
	Beta	0.6	0.6	1.7	1.7	0.7	1.2	1.4	1.5
2	D-prime	5.1	3.0	3.6	2.0	5.2	2.7	4.8	2.6
	Hits	95.3	4.9	91.2	8.5	94.5	9.6	95.3	5.7
	FA's	7.2	7.9	11.2	8.4	6.5	7.1	11.6	13.4
	Beta	1.3	1.5	1.2	1.5	1.0	1.2	1.1	1.6

Days: 1 = normal sleep; 2 = sleep deprived;
X = mean score; s_x = standard deviation;
EX = pre-task cycle exercise; NEX = no exercise;
Hits = percent hits; FA's = percent false alarms.
Atropine or placebo was administered between task cycles 1 and 2.

TABLE 3

Atropine and Sleep Deprivation Effects on Auditory Vigilance

		Atropine		Placebo	
Task Cycle		Day 1	Day 2	Day 1	Day 2
1	D-prime	5.46	4.24	5.76	4.04
	Hits	87.0	78.7	89.0	76.6
	FA's	1.08	1.55	1.06	1.56
	Beta	3.76	4.05	3.72	4.40
	RT	538	530	549	572
2	D-prime	4.37	3.19	5.16	3.66
	Hits	80.9	66.6	87.0	73.8
	FA's	1.76	2.39	1.58	2.24
	Beta	4.01	4.22	3.80	4.31
	RT	537	553	545	580

Response measure = mean scores
Day 1 = normal sleep; Day 2 = no sleep.
Hits = percent hits; FA's = percent false alarms
Atropine or placebo was administered between task cycles 1 and 2.

This effect probably depends on decrements in sustained attention, rather than increased caution, the typical explanation for increased beta scores. This interpretation is supported by increased hits RT variability ($p < .0001$), without increased mean RT's for hits ($p < .4$), combined with increases in both mean RT ($p < .0001$) and RT variability ($p < .0001$) for false alarms. This is the typical pattern of response changes after sleep deprivation, i.e., subjects who have experienced lapses in attention try to correct their performance on self-perceived missed trials. This produces less accurate, but more lengthy and variable performance, particularly for errors of commission. Time-on-task reduced d-prime ($p < .001$) and hits ($p < .0001$), and increased beta scores ($p < .0001$). Its interaction with sleep deprivation further reduced d-prime ($p < .009$) and hits ($p < .006$). Sleep deprivation and exercise interactions also reduced d-prime ($p < .003$) and hits ($p < .03$) and increased beta scores ($p < .005$), (Table 4). These data reflect the profound decrements in perceptual acumen and vigilance caused, particularly, by sleep deprivation, and combined with the data from the aircraft signal detection task, indicate that both atropine and sleep deprivation produce general reductions in afferent perceptual information processing. In addition, a full night of sleep deprivation was shown to have additional adverse effects on the sustained deployment of attention which extend beyond the effects produced by a 2.0 mg dose of atropine.

TABLE 4

Atropine, Sleep Deprivation, Exercise and Time on Task Effects on Auditory Vigilance D-prime

ATROPINE

		EX				NEX			
		Day 1		Day 2		Day 1		Day 2	
Task Cycle	Block	X	s_X	X	s_X	X	s_X	X	s_X
1	1	5.6	2.9	4.5	2.4	6.3	2.9	6.1	2.8
	2	6.1	2.8	3.7	1.4	6.4	2.4	4.7	1.8
	3	5.3	2.4	3.1	1.2	4.8	1.7	4.6	2.0
	4	5.2	2.7	3.0	1.4	5.5	2.3	4.9	1.7
	5	5.5	2.6	3.1	1.4	5.0	1.8	4.7	1.8
	6	4.6	2.4	3.9	2.3	5.2	1.5	4.5	1.5
	X	**5.4**	**2.5**	**3.6**	**1.7**	**5.6**	**2.1**	**4.9**	**1.9**
2	1	4.6	2.3	3.6	1.7	5.3	2.4	3.9	2.3
	2	3.8	2.3	2.6	1.1	4.6	2.1	3.3	1.6
	3	4.4	2.5	3.2	1.8	5.1	2.3	4.1	1.8
	4	4.2	2.2	2.5	1.1	3.7	2.0	3.6	1.5
	5	3.9	1.9	2.5	1.0	4.5	2.5	3.2	2.1
	6	4.1	2.0	2.6	1.5	4.2	1.6	3.2	1.4
	X	**4.2**	**2.2**	**2.8**	**1.4**	**4.6**	**2.2**	**3.6**	**1.8**

PLACEBO

Task Cycle	Block	X	s_X	X	s_X	X	s_X	X	s_X
1	1	6.7	2.9	4.3	2.1	5.5	2.9	4.8	2.6
	2	6.6	2.8	4.2	2.2	6.3	2.4	4.1	1.5
	3	5.4	2.3	3.6	1.6	5.1	2.0	4.0	1.9
	4	6.6	2.7	3.4	1.8	4.9	2.3	3.9	2.0
	5	6.2	2.4	4.2	1.8	5.3	2.4	3.7	1.4
	6	5.3	2.1	4.0	1.5	5.2	2.2	4.2	2.2
	X	**6.1**	**2.5**	**4.0**	**1.8**	**5.4**	**2.4**	**4.1**	**1.9**
2	1	6.1	2.9	4.4	2.3	5.5	2.3	4.1	1.7
	2	5.9	2.7	3.5	1.6	4.8	2.4	3.8	1.8
	3	5.2	2.9	3.5	1.7	4.9	1.8	3.4	1.6
	4	5.3	2.6	3.6	1.6	4.8	2.2	3.6	1.9
	5	4.5	2.3	3.1	1.4	4.8	2.0	4.2	2.2
	6	4.9	2.1	3.1	1.4	5.2	2.0	3.7	1.8
	X	**5.3**	**2.6**	**3.5**	**1.7**	**5.0**	**2.1**	**3.8**	**1.8**

Days: 1 = normal; 2 = sleep deprived.
EX = pre-cycle exercise; NEX = no exercise.
X = mean score; s_X = standard deviation; Block (1-6) = time on task.
Drug dose given between task cycles —

PHYSIOLOGICAL RESULTS

The physiological variables, *Multiple Sleep Latency Test,* and self report measures provided confirmation of treatment effects on autonomic and arousal mechanisms. These effects could not be related specifically to the performance decrements seen, as they were state measures intended to describe differential autonomic and arousal profiles generally underlying performance. The combined results of these measures indicate that discriminative profiles of such measures can be obtained which enhance the understanding of the treatment effects discovered, possibly within the context of the "notational multidimensional arousal space" thought to subserve performance (Hamilton, et al., 1977).

Heart Rate

The 2.0 mg atropine dose caused a significant ($p < .001$) heart rate increase, from a mean of 68 BPM to 100 BPM, by the seventh measurement session of the day, which returned to 75 BPM by the last (eighth) recording session of the day. Sleep deprivation had no main effects on heart rate, nor were there any (atropine x sleep deprivation) interaction effects. The main effects of exercise on heart rate ($p < .0001$) were greater than those of atropine, although the (atropine x exercise) interaction effect was not significant. The rates were elevated from a baseline of about 70 BPM to 152 BPM after exercise, returning to a mean of 84 BPM by the next recording session. An underadditive interaction between exercise and sleep deprivation ($p < .02$) was also found, where heart rates were initially higher after sleep deprivation, but increased less after exercise on that day (see Table 5).

Blood Pressure

Atropine had no significant effects on systolic pressure, but diastolic pressure was increased ($p < .0001$), producing increases in mean arterial pressure ($p < .002$). Sleep deprivation had no effects on any blood pressure measure, either alone or in combination with the other stressors. Exercise affected systolic ($p < .0001$), diastolic ($p < .0007$) and mean arterial ($p < .002$) pressures, reflecting the physiological changes necessary to support the exercise workload. However, there were no interactions with atropine (see Table 6).

Pupil Diameter

An atropine-produced increase in pupil size ($p < .0001$) persisted throughout the experimental day after atropine administration, except where the main effect of exercise ($p < .04$) counteracted this effect (Table 7). However, this effect was not large enough to establish an (atropine x exercise) underadditive interaction ($p < 1$).

TABLE 5

Atropine, Sleep Deprivation and Exercise Effects on Heart Rate

	Exercise		No Exercise	
Day	Atropine	Placebo	Atropine	Placebo
1	70	70	70	68
	152	170	67	69
	82	89	67	69
	62	73	65	67
	75	77	68	68
	155	156	69	156
*	108	81	99	69
	76	68	76	66
2	73	72	74	69
	147	144	73	68
	80	84	72	70
	61	67	66	67
	76	79	74	69
	154	151	73	70
*	107	77	101	70
	77	66	74	67

(*) = Time of atropine injection
Day 1 = normal sleep; Day 2 = No sleep;
Measurements bracketed exercise (1&2,5&6); and task cycle

TABLE 6

Atropine, Sleep Deprivation and Exercise Effects on Blood Pressure

	Exercise				No Exercise			
	Atropine		Placebo		Atropine		Placebo	
Day	SBP	DBP	SBP	DBP	SBP	DBP	SBP	DBP
1	132	79	130	81	139	81	140	80
	139	75	143	81	136	77	135	80
	128	75	129	77	134	78	137	77
	125	75	127	77	134	82	134	81
	132	81	132	79	142	79	143	80
	139	76	142	78	137	79	138	80
*	128	78	125	73	138	84	137	77
	125	78	123	74	130	80	137	81
2	128	77	134	80	136	81	140	79
	137	79	141	81	131	75	137	80
	131	73	130	79	135	77	134	77
	128	73	127	77	134	78	133	79
	134	80	129	78	141	79	140	79
	139	80	140	81	136	78	137	77
*	135	78	133	71	135	83	134	76
	124	77	131	79	129	78	134	83

(*) = Time of atropine injection
Day 1 = normal sleep; Day 2 = No sleep;
SBP = Systolic pressure; DBP = Diastolic pressure
Measurements (1&2,5&6) bracketed exercise; and task cycle

Sleep Onset Latency

The means for the MSLT revealed decreased sleep onset latencies after all treatments. A four way (drug x day x cycle x exercise) ANOVA found main effects of atropine ($p < .0001$) and sleep deprivation ($p < .0001$), as well as an (atropine x sleep deprivation) hyperadditive interaction effect ($p < .004$). While there was no main effect of exercise or significant second-order interaction between exercise and either atropine or sleep deprivation, a third-order interaction among all three stressors was evidenced, where the combined effects of atropine and sleep deprivation were made worse after exercise ($p < .03$). This effect confirms severe reductions in arousal caused by combination of these three stressors (see Table 8).

TABLE 7

Atropine, Sleep Deprivation and Exercise Effects on Pupil Diameter

	Exercise		No Exercise	
Day	Atropine	Placebo	Atropine	Placebo
1	3.5	3.2	3.6	3.4
	3.4	3.1	3.4	2.9
*	3.8	3.3	4.0	3.4
	3.9	3.1	4.3	3.2
2	3.4	3.3	3.3	3.3
	3.5	3.2	3.2	3.2
*	3.6	3.2	3.7	3.5
	4.1	3.0	4.2	3.2

(*) = Time of atropine injection
Day 1 = normal sleep; Day 2 = No sleep;
Measurements bracketed each task cycle

TABLE 8

Atropine, Sleep Deprivation and Exercise Effects on Sleep Onset Laten .y

	Exercise		No Exercise	
Day	Atropine	Placebo	Atropine	Placebo
1	12.3	12.5	10.7	12.1
*	5.6	10.0	6.0	13.3
	3.9	12.3	6.6	13.5
2	4.1	4.3	4.6	6.2
*	2.1	4.1	3.8	5.0
	1.3	3.7	3.1	5.6

(*) = Time of atropine injection
Day 1 = normal sleep; Day 2 = No sleep;
Measurements taken just prior to task cycle (after exercise) and at day's end.

Self Reports

The effects of atropine resulted in perceived decreases in alertness, competence, and attentiveness, whereas the effects of exercise appeared related more to comfort and motivation. Sleep deprivation effects were pervasive, and may reflect subject-perceived "demand characteristics" rather than actual stress effects, as almost all of the items displayed significant changes toward reduced functioning. (Table 9).

DISCUSSION

Performance Tasks

Atropine was shown to produce exclusive reductions in perceptual input processing functions in both performance tasks, without causing changes in responsiveness. The effects of sleep deprivation on afferent functions generally paralleled those found for atropine. Decreases in stimulus sensitivity and correct detections were found in both the aircraft identification and auditory vigilance tasks, although decrements in responsiveness were also found in task cycle 1 in the visual task and in both task cycles for the auditory vigilance task after sleep deprivation. While this effect on responsiveness could signal an increased caution toward responding as responsible for the decline in d-prime and hits, the decreased sleep onset latencies found with the MSLT, in particular, suggest that these changes are more likely due to lapses in attention resulting from lowered arousal, i.e., sleepiness. Thus, subjects failed to respond as readily or as accurately because they failed to acquire the necessary information. The interactions of atropine and sleep deprivation which produced further reductions in perceptual sensitivity without further effects on response bias in the visual and auditory tasks supports this contention, especially when compared to the interactions of sleep loss with time-on-task and exercise, which did reduce responsiveness to further impair performance in the auditory vigilance task. This suggests that when exercise was performed, fatigue, rather than the expected physiological activation, was the net effect.

TABLE 9
ATROPINE, SLEEP DEPRIVATION & EXERCISE
EFFECTS ON SELF REPORTS

	Atropine	Sleep Loss	Exercise	
ACTIVE	.0012	.0001		PASSIVE
CONFUSED	.0230	.0001	.05	CLEAR THINKING
COLD			.0065	HOT
AWKWARD	.0001	.0001	.0001	COORDINATED
ENTHUSIASTIC	.0375	.0001		BORED
WORRIED		.0001		CONFIDENT
EXCITED		.0004		CALM
ALERT	.0409	.0001		DROWSY
ENERGETIC	.0062	.0001		LETHARGIC
EFFICIENT	.0036	.0001		INEFPICIENT
APATHETIC		.0001	.0006	INTERESTED
SAD		.0001	.0186	HAPPY
DREAMY	.0163	.0001	.0163	ATTENTIVE
CLEAR VISION	.0001	.0001		BLURRED VISION
DIZZY	.0001	.0001	.0011	STEADY
INVOLVED	.0001	.0046		UNINVOLVED
SUSPICIOUS		.0130		TRUSTING
IMPATIENT		.0003		PATIENT
DULL		.0001		SHARP
STRONG	.0036	.0001	.0003	WEAK
FAST	.0268	.0001	.0325	SLOW
AWAKE		.0001		SLEEPY
HOSTILE		.0001		FRIENDLY
HEALTHY		.0001	.0003	SICK
SOBER	.0003	.0001	.0011	DRUNk
COMFORTABLE		.0001		UNCOMFORTABLE
REFRESHED		.0001		WEARY
SWEATY			.0001	DRY

Response measure = significance of effect;
Self-report measurements bracketed each exercise and task cycle

Physiological Variables

The data on cardiovascular function provided the expected changes relative to stressor effects. Heart rate changes secondary to atropine administration displayed the usual biphasic time course related to parasympathetic blockade, which was exacerbated only slightly by exercise. However, exercise had the most dramatic effects, increasing heart rates two-fold over baseline values. Sleep deprivation caused a reduction in this exercise effect, probably because of decreased physiological reactivity, without producing main effects of its own.

Systolic blood pressure displayed no systematic changes; blood pressure changes were limited to atropine effects on diastolic pressure, leading to similar changes in mean arterial pressure. This combination of effects is what has generally been found, and were coincident with the effects seen on perceptual sensitivity. Exercise produced effects on all blood pressure measures consistent with the changes in heart rate needed to support the exercise workload. These effects were apparently not related to performance decrements seen after combination of exercise with sleep deprivation, as the exercise by sleep deprivation interaction effect failed to approach significance for either of the blood pressure measures when performance was significantly impaired. Exercise had no interactions with atropine on any blood pressure measure, nor did sleep loss. Thus, the ability of cardiovascular function, as described here, to provide insights about arousal mechanisms underlying performance appears small, and would be limited to changes in diastolic pressure and their effects on cardiac output. The ability of such variables to ultimately describe performance mechanisms will likely be more closely linked to real-time changes in the variability of cardiovascular function.

The effects of atropine on pupil size were highly significant. Enlargement of pupils continued throughout the measurement periods, except where sympathetic activation produced by exercise was able to counteract this effect slightly. This exercise effect was not significant, however, nor were there effects of sleep deprivation on pupil sizes. Although it could be argued that these peripheral pupillary effects are responsible for the visual performance decrements found, the effects found on perceptual encoding in the auditory task which paralleled these pupillary changes support an interpretation of centrally-mediated arousal-based effects.

The sleep onset latency scores provided the greatest indications of impairments in arousal as being responsible for performance decrements. Main effects of decreased sleep onset time were found for atropine, as well as sleep deprivation, and the combination of these two stressors reduced sleep latency further. When combined with the independent and interactive effects of these two treatments on perceptual encoding, reductions in sleep onset latency point strongly toward reduced arousal as the mediating mechanism responsible for input processing deficits.

The self reports generally supported these conclusions. Atropine scores reflected widespread self-perceived decreases in competence and attentiveness, while exercise effects were confined almost entirely to variables indexing decreased vigilance or autonomic results of physical activity, e.g., sweaty, hot. These effects also paralleled the MSLT results, as well as the performance effects. The sleep deprivation effects on self reports were disappointing, however, as the widespread changes reported after sleep loss indicated a generalized lack of motivation or perhaps demand characteristics inadvertently instilled in the subjects. In combination, however, the responses suggest that properly administered questions about self-perceptions of subject state can provide valuable validation of arousal-mediated effects on performance and autonomic functions

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SUMMARY

In summary, the results attest to the common disruptions in perceptual sensitivity produced by atropine and sleep deprivation, probably through related, but different effects on arousal. This interpretation is supported both by the synergistic interaction effects of these two stressors on discrete information processing activities, as well as their common effects on general arousal as assessed by the MSLT. The dearousing effects of a full night of sleep deprivation extend beyond those of 2.0 mg of atropine, however, indicating either that sleep deprivation has

effects on additional neural mechanisms supporting more effortful activities, or that the dose of atropine required to produce such effects is greater than 2.0 mg. The possible effects found for sleep deprivation on response output functions could support the former argument, although other studies conducted with larger atropine doses have found effects similar to those seen here for sleep deprivation. Only additional investigations using larger atropine doses with tasks similar to those used here could fully answer this question. No main effects of prior moderate exercise were displayed for any performance measure, including sleep onset latency. This situation indicates minimal effects on arousal, beyond those produced while exercising, except where the combination of exercise and the other stressors was shown to produce fatigue. Although this compromises the common notion that exercise might produce extended enhancements in perceptual encoding through extended physiological activation (see Tomporawski & Ellis, 1986), it strengthens arguments that the common, but discriminative, adverse effects of atropine and sleep deprivation are produced through similar adverse effects on arousal (mechanisms). Further study into the real-time changes in arousal-controlled performance and physiological functions should provide more discriminative functions by which to signal imminent decrements in human performance, leading to practical applications in which human performance may be accurately predicted via physiological measurement.

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